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SURGICAL CORRECTION OF AORTIC INSUFFICIENCY*

The major causes of insufficiency of the aortic valve are rheumatic fever, syphilis, subacute bacterial endocarditis, trauma, anomalies of the aortic valve associated with one of the above or another anomaly such as coarctation, and hypertensive cardiovascular disease (relative insufficiency).

Free aortic insufficiency may exist as a single, relatively pure lesion or in combination with other valvular lesions. In the past the high incidence of syphilis and luetic cardiovascular disease was closely associated with free aortic insufficiency. With the decreasing incidence of untreated or inadequately treated syphilis there has been a marked decrease in aortic insufficiency due to this cause in most areas of this country. However, free aortic insufficiency due to rheumatic valvulitis remains a common lesion. It frequently exists as the primary lesion without major degrees of aortic stenosis or mitral stenosis. When the disease is of rheumatic origin, there has been in the past frequent confusion of the lesions by auscultation, particularly in the later phases of the disease.

When free aortic insufficiency has existed for prolonged periods of time, it is frequently associated with a rumbling diastolic murmur which may be extremely difficult to differentiate from the murmur of mitral stenosis. The dilatation of the left ventricle may give rise in addition to a loud systolic murmur of mitral insufficiency. At autopsy or operation it will be found frequently that no specific organic lesion of the mitral valve itself is present but merely dilatation of the mitral valve annulus with a relative type of insufficiency. Not uncommonly there will also be a systolic murmur over the aortic area which is not necessarily due to major degrees of aortic stenosis but which may be due to dilatation of the aorta just distal to the valve and to a relative stenosis. Therefore, it is important to keep these factors in mind in attempting to evaluate the primary lesion.

The clinical course of aortic insufficiency of the slowly progressive type differs sharply from those types of aortic insufficiency in which sudden valvular incompetence occurs. In luetic heart disease disruption of a cusp may occur. In acute or subacute bacterial endocarditis a major deformity of one or more cusps may occur rapidly, and in acute trauma the rupture of the valve occurs instantly. In these groups failure may occur within days to weeks after the onset of the lesion, and the overall prognosis after the onset of the lesion is extremely short. In the luetic type in which the major cusp deformity has occurred and following acute trauma, total life expectancy can be estimated as averaging from six to twelve months. In acute and subacute endocarditis the prognosis is not so certain, and the period of survival depends primarily upon the degree of deformity which has been produced. In some cases it should be noted that the rapid type of course may ensue.

In aortic insufficiency of rheumatic origin the usual course is one of slow progression with gradual cardiac enlargement leading ultimately to signs of failure and frequently associated with angina pectoris. In such cases the angina is not necessarily related directly to exertion but often is nocturnal in type. It has been postulated that angina pectoris occurs primarily with luetic aortic insufficiency and is related to the narrowing of the orifices of the coronary arteries. We feel that this is not true in the majority of cases but is due to an inadequate coronary flow in relation to the increased cardiac work. In pure aortic insufficiency major cardiac enlargement usually precedes the onset of serious symptoms. Thus, the patient may have progressed to a point of severe myocardial damage before he seeks medical aid.

The occurrence of auricular fibrillation is not rare in the later phases of the disease even without mitral lesions, and ventricular conduction abnormalities and ventricular arrhythmias are common. Many patients with this type of lesion die suddenly, apparently from ventricular fibrillation with instant death.

Medical management of aortic insufficiency has always offered serious problems once evidences of congestive failure have been made manifest. Since the mechanical element plays such a large role in the development of failure, it would appear reasonable that, if this defect could be reduced or be completely corrected, the

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patient's outlook would be greatly altered. Because of the severe myocardial changes which occur concomitantly with great cardiac enlargement, it is apparent that ideally such correction of the valvular defect should be instituted early. If such correction is carried out at a later date, it is not entirely predictable as to the number of changes which are reversible. As in the treatment of all valvular disease, surgery essentially offers a mechanical correction of a mechanical lesion.

Many attempts have been made to reconstruct the aortic valve. These early attempts have consisted chiefly in the transplantation of tissue in an effort to occlude the leaking portion of the aortic orifice. While some of these attempts have shown promise in the early phases, the late results have been less satisfactory than had been anticipated. More recently the use of plastic materials, either as cloth ore rubber-like substitutes, have been used in an effort to form a valve. Similarly these materials have not entirely lived up to their early promise.

Since 1944 we have been engaged in the investigation of this problem and have developed a plastic aortic valvular prosthesis which has been used clinically for the correction of this serious lesion. The method which we have employed for the past three years has been the insertion of a ball-valve into the distal portion of the aortic arch. The prosthesis is held in place by nylon rings which do not necrose the aorta because of multiple point fixation. Early in our experiences with this prosthesis peripheral embolization occurred in some cases. This was not due to clotting within the valve itself but was due to clotting which formed at the prosthesis-aortic junction, apparently because of angulation of the prosthesis or a discrepancy between the size of the prosthesis and the aorta. We now utilize a small cuff of orlon mesh to insure proper fit and remove a segment of the aorta so that angulation does not occur. This has eliminated the problem of embolization. The prosthesis has been placed into the distal aortic arch because at this level the aorta can be occluded safely without serious cardiac embarrassment and without the necessity of interruption of venous flow to the heart. Actual insertion of the valve itself requires only three to five minutes; while the safe period of occlusion at this level is at least twenty minutes. This makes for a simpler procedure, which patients with extremely severe lesions can tolerate when they cannot withstand major manipulations at the aortic root in the region of the aortic valve. When mitral stenosis or aortic stenosis is present, it may also be corrected during the same operation.

Since 1952 we have had the opportunity of observing a large number of patients with aortic insufficiency. Ninety such patients have had this type of operation for aortic insufficiency. Until recently we have not refused operation to patients because of the terminal stage of their disease, but we did elect to postpone operation in patients whose course had not been positively shown to be progressive. Essentially all patients have shown signs of congestive failure. All have had a diastolic gallop rhythm, and many have shown rumbling diastolic murmurs at the apex. Major cardiac enlargement has always been present. All of the patients selected have had extremely wide pulse pressures and diastolic pressures of 40 mm. of mercury or less. A majority of the patients have had severe angina pectoris, and secondary liver disease due to prolonged congestion has been common. The cases have been divided among all of the etiologic agents with a dominance of rheumatic lesions.

As we have gained more experience, we have felt that the indications for operation in an ideal patient should be outlined as follows: (1) preferably under 50 years of age; (2) relatively pure aortic insufficiency or with a second correctible lesion; (3) absence of acute rheumatic activity; (4) signs of progression on medical management or with controllable failure without overt congestic failure; (5) cardiac enlargement of only moderate degree; (6) absence of intractable angina pectoris; (7) no serious changes in ventricular conduction; (8) absence of serious renal or hepatic disease. It is quite apparent that many of the patients who require operation will not fall into this group, but these criteria represent the ideal patient who will obtain maximum arrest of his disease with minimum risk.

We feel that rheumatic patients in whom the acute onset of failure may occur without evidence of a sudden change in their mechanical lesion frequently have severe rheumatic myocarditis and are having an exacerbation of their rheumatic process. We strongly advise against operation in such patients since it has been our experience that they tolerate operation extremely poorly. We also believe that patients who have extreme cardiac dilatation and who are over the age of 50 do not do well with this operation. Whether this cardiac dilitation is associated with impairment of the coronary flow by intrinsic coronary disease or whether it is also a function of loss of myocardial reserve after a prolonged disease is not certain.

In patients who have primarily hypertensive cardiovascular disease one may see an elevation of systolic pressure with a normal or elevated diastolic pressure. In these patients there may be a considerable widening of the pulse pressure and a typical murmur of aortic insufficiency. We do not believe that such patients are suitable for operation at this time, since further elevation of the diastolic pressure would seem to serve no useful purpose. Aortic insufficiency of this type is relative, and it does not carry the same prognosis as does free aortic insufficiency with major lowering of the diastolic pressure.

The frequent occurrence of serious ventricular arrhythmias in the later stages of all types of free aortic insufficiency makes anesthesia and operation a serious problem. The development of these arrhythmias is completely unpredictable and extremely difficult to control. A number of patients who have been accepted for operation have died while in the hospital prior to operation while undergoing excellent medical management. It must be remembered that, while operation may correct the valvular defect, it does not immediately effect the ventricular conduction system.

The first operation involving the insertion of a plastic valvular prosthesis was carried out in September of 1952, and the first patient is now working daily, after having been in failure prior to operation. Operative mortality rate has been steadily diminishing primarily because of selection of patients earlier in the course of their disease. Operation has little to offer the patient who is already terminal because of his myocardial changes. It is the patient in the early to middle classifications that operation offers real benefit. Essentially all patients in these groups have shown marked improvement with restoration of their ability to carry out all normal adult activities. Patients who have shown cardiac dilatation preoperatively have shown considerable diminution of cardiac size postoperatively. Those who show only moderate cardiac enlargement may show moderate or no decrease in size. We have encountered no case in which it was not possible to insert the valve at operation. There has been one case in which disruption of the aorta occurred some months after operation due to pressure upon the aorta. In the relatively ideal patient mortality rates should approximate 10 per cent; while in the very advanced patients mortality rates approximate 40 per cent. This much higher mortality rate in the far advanced patients is also found when one deals with other valvular lesions. It must be borne in mind, however, that aortic insufficiency is a much more malignant lesion than mitral stenosis even though early in its course its manifestations may appear relatively innocuous.

Currently other procedures are under investigation which may offer more benefit than the present procedure by controlling all of the reflux of all the insufficiency, and changes in the type of valve currently being used may tend to eliminate the sound which the valve now makes. The sound of the valve has not been a problem in the patients we have observed, and this sound tends to diminish after the valve becomes surrounded by fibrous tissue. It has been remarkable that no patient has felt that the sound made by the valve has constituted a problem to him; apparently the general increased sense of well-being and ability to work has been so striking that the patient soon completely overlooks the sound factor.

SUMMARY

Patients with free aortic insufficiency show wide pulse pressure and low diastolic pressures. Those who show acute onset of this lesion should be considered for operation early. Signs of early congestive failure, increasing heart size, or progression of symptoms also merit consideration for operation. Extreme cardiomegaly, acute rheumatic fever, age over 50, endocarditis, and the presence of ventricular arrhythmias increase operative risk.

Operation is not advocated in asymptomatic patients.

Operation is advised when signs or symptoms of progression are present and before such extensive myocardial damage has occurred as may seriously limit the result.

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The opinions and conclusions expressed herein are those of the author and do not necessarily represent the official views of the Scientific Council of the American Heart Association.

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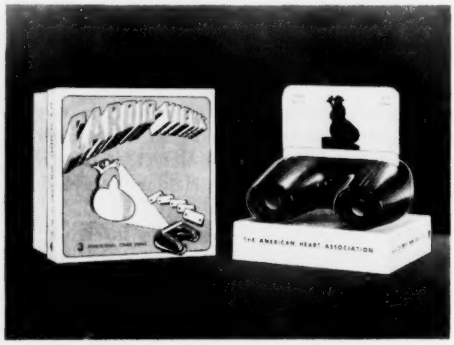

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